EFFECT OF FEMALE SEX HORMONES ON AGE RESISTANCE OF CHICKENS TO PARASITISM

by

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INTRODUCTION AND REVIEW OF LITERATURE

Resistance of animals to parasitism which has come into greater prominence during the last three decades has three main subdivisions, natural resistance, acquired resistance and age resistance. Natural resistance to parasitism is generally understood to mean the resistance of an animal that is normally present. Acquired resistance may be defined as the resistance exhibited by an animal to parasitism due to previous or contemporary infection. Age resistance is the resistance of an animal to parasitism which develops as the animal grows older, especially to maturity.

In studying resistance, Chandler (1932a) held that two factors had to be considered. He determined these to be the resistance of the host to further infection and the host's resistance to the effects of parasitism. Several standards have been used as bases for measuring the degree of resistance of the host to the effects of parasitism. Herrick (1926) used as a basis the comparative rate of growth of parasites in hosts of different ages. Ackert and Herrick (1928) evaluated the amount of resistance by comparing the numbers of parasites that were able to live in hosts and the effects of parasites on young and old hosts. Chandler (1932) used as a basis the resistance of a host to reinfection. In 1936, Stoll found that a measurement of resistance of the host could be made by comparing the development of the worms and their egg-laying ability.

In studies made by Ackert, Fisher and Zimmerman (1927) and Ackert, McIlvaine and Crawford (1931), it was found that diets deficient in vitamin A lowered the resistance of hosts to helminthic infections. Zimmerman, Vincent and Ackert (1926) and Ackert and Nolf (1931) found that the resistance of hosts was lowered by diets deficient in vitamin B. The accounts of this work with vitamins A and B appear to be the first conclusive evidence that natural resistance of hosts to helminthic infection may be lowered by nutritional deficiencies. Studies on vitamin D in relation to resistance were carried out by Ackert and Spindler (1929). Additional researches in dietary supplements as factors in resistance were done by Ackert (1927), Ackert and Beach (1933), Ackert, Branson and Ameel (1944), Ackert and Riedel (1946), and Riedel (1949).

Comparisons of resistance of hosts were made by Ackert, Eisenbrandt, Wilmoth, Glading and Pratt (1935). They found significant differences in the natural resistance of respective heavy and light breeds of chickens. Barred Plymouth Rocks and White Plymouth Rocks, as heavy breeds of chickens, had fewer and smaller _Ascaridia lineata_[1] than did the light breeds, White Leghorn and White Minorca.

A review of these and other phases of resistance was made by Taliaferro (1929), La Page (1939) and Ackert (1942).

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[1] This nematode is known also as _Ascaridia perspicillum_ and as _Ascaridia galli_.

Of these different types of resistance exhibited by animals to helminthic infection perhaps the most important is age resistance. As stated, it does not arise as a result of previous or contemporary infection but develops as the host grows up to approximate sexual maturity.

Increased resistance of animals to parasitism was first noticed by Looss (1911). He found that hookworm larvae fed to young dogs were able to reach maturity; however, when they were fed to adult dogs the results were negative. In 1920 Ransom and Foster confirmed Looss's findings with *Ascaris* in pigs. Ransom (1921) demonstrated age resistance in chickens to the nematode *Syngamous trachea*. Herrick (1926) found a gradual increase in the resistance of chickens as they grow older to the development of the nematode *Ascaridia perspicillum*. He found that the resistance increased with age up to 103 days. In 1928, Ackert and Herrick reported additional evidence of this kind. In their experiments using chickens they showed that the percentage of infections ranged from 85.7 per cent to 100 per cent in experiments terminated at the end of three weeks. The infection ranged from 33.4 per cent to 66.7 per cent in experiments terminated at 8 to 45 weeks.

Age resistance was demonstrated by Sarles (1929) who infected young and adult dogs with *Ancylostoma braziliense*. Herrick (1928) found that older dogs were more resistant to *Ancylostoma caninum* than younger ones. He showed that it took the hookworms longer to mature in older dogs and that the proportion of larvae which developed decreased greatly with age. Experimental work by Scott (1928) and McCoy (1931) resulted in confirmation of Herrick's work.

Africa (1931) demonstrated age resistance in laboratory rats against *Nippostrongylus muris*. Results of experiments on this helminth by Chandler (1932) were not as striking as those of Africa. Winfield (1933) noted a marked increase in resistance in older rats to the nematode *Heterakis spumosa*.

Age resistance in animals has been observed also to flatworm parasites. It was shown by Shorb (1933) that age resistance manifests itself in the rat and the mouse against the cestode parasite *Hymenolepis fraterna*. In studies with flukes, Anzel (1934) noticed an increased resistance in older snails to larval forms. Ackert and Reid (1937) showed a marked increase in resistance in older chickens to the cestode *Raillietina cesticillus*.

Ackert, Porter and Beach (1935) demonstrated age resistance in chickens to the growth of *Ascaridia lineata*. They found that the maximum resistance is ordinarily reached when the chicken has attained the age of 93 days. Age resistance was demonstrated in mice to the nematode *Trichinella spiralis* by Riedel (1948). He found that mice infected when about six weeks of age were much less resistant than mice infected when more than five months of age. Hendricks (1943) presented quite striking results using the same host and parasite. He found that after administering 50 larvae the young mice showed an average of 75 per cent development of adult worms compared with 28.4 per cent development for the old mice.

It has been suggested that secretions of one or more of the ductless endocrine glands may play a role in the resistance of animals to parasites. However, it was found by Ackert (1924) that the removal of the chicken thymus gland had no effect on the growth of *Ascaridia*. Jaffe (1926) showed that the resistance of rats to parasitism was reduced by suprarenalectomy. Ackert and Otto (1927) noted that the size of the chicken thyroid gland was not changed due to parasitism with *Ascaridia lineata*.

A possible relationship between the products of the sex glands and resistance to parasitism was found by Stoll (1936). He observed that the resistance of a ram to the nematode *Haemonchus contortus* was reduced during the breeding season. Gunther (1942) suggested that there are many "differences in sexual incidence of parasitism, especially in worm infections." He noted that there was a distinct difference in the number and kind of parasites harbored by male and female adult individuals, but that there were no similar differences found in children which are known to be susceptible to many helminths. It was his contention that "hormonal secretions may have an influence on certain parasites."

Addis (1946) suggested some relationship between sex hormones of the host and the vitamin G complex due to the fact that the growth of *Hymenolepis diminuta* is normal in male rats on a vitamin-deficient diet, while it is stunted in female rats on the same diet. Sadun (1948) reported that moderate doses of testosterone propionate to immature male chickens and of alpha estradiol benzoate to immature female chickens increased their resistance to infections with _Ascaridia galli_. Todd (1949) found that there were no significant differences in per cent of development of _A. galli_ and _Heterakis gallinae_ in mildly hyperthyroid, mildly hypothyroid, and normal fowl hosts. Ackert and some of his students noted that the resistance of chickens to parasitism increased as they approached sexual maturity.

The foregoing suggested relationships between sex glands and degree of parasitism and the increase in resistance of fowls to the nematode _A. galli_ that has been noted up to approximate sexual maturity led to the present study. The object of the investigation was to ascertain if injections of the female sex hormone, diethylstilbestrol, into young pullets would increase their resistance to the fowl nematode _A. galli_.

MATERIALS AND METHODS

The chickens used in these experiments were Single Comb, White Leghorn pullets obtained as day old chicks from a commercial hatchery. Upon arrival the chicks were banded, divided into groups and put into electrically heated battery brooders. The chicks received a commercial food containing all essential ingredients in the proper proportion. Grit and water were accessible to them at all times.

At the age of one week, the chicks were divided into two groups of approximately equal weight. In all experiments the birds in Group I received the injections of female hormone while those of Group II were maintained as a control group.

The diethylstilbestrol was obtained commercially in a crystalline form. This was used in the proportion of 10 mg of the hormone to one cc of Mazola oil. It was necessary to warm the mixture slightly to cause the hormone to go into solution.

When the chicks were 12 days old, injections of the female hormone thus prepared were administered into the pectoralis major muscle at the rate of 0.1 cc per chick. The hormone was given every second day excluding Sundays until the chicks had received 10 injections. In all, each chick received a total of 10 mg of diethylstilbestrol.

Two days after the initial injection of hormone, each chick of both groups was parasitized with equal numbers of embryonated, viable _A. galli_ eggs. The eggs were obtained from adult female worms obtained from packing companies. Upon arrival the worms were dissected and the two uteri were removed, but kept intact and placed in water in a petri dish. The water depth was kept at approximately four millimeters. The eggs were incubated at 20° C. During the incubation period, the culture was agitated periodically as an aid in supplying oxygen to the eggs.

The culture was incubated about 30 days to enable the larvae in the eggs to develop and pass through the first molt, at which time they become infective.

Each chick of both groups was parasitized by the drop method of Riedel (1947). In this method about one-half inch of clean sand is placed in a dropping bottle. The egg culture is poured into the bottle and sufficient water added to cover the sand one to one and one-half inches. The bottle is shaked vigorously to break up the uteri and free the eggs from albumen or other material which causes them to clump together. This prepared culture is agitated to give a uniform suspension of eggs per drop of the mixture.

The process of standardizing the suspension was then undertaken. A drop of the suspension was placed on a glass slide and the viable, embryonated eggs were counted. A series of drops was counted in this manner and the average determined. The concentration of the suspension was adjusted to the desired point by the addition of water and again standardized. When the correct concentration was reached the chicks were parasitized with the proper number of drops which would make a total of about 200 _A. galli_ eggs. The procedure followed was to open the chick's mouth and allow the drops to fall well back in its throat. It was necessary to agitate the suspension between each feeding to insure a uniform suspension.

Three weeks after parasitizing the chicks were killed. The intestine from the gizzard to the yolk sac diverticulum was removed immediately and flushed into a glass jar by the hydraulic method of Ackert and Wolf (1929). After the worms had been left in the jars several hours to lose their motility, they were killed and preserved in 10 per cent formalin. The formalin also aided in preventing coiling of the larvae and the absorption of water sufficient to rupture the worm's body wall.

The worms were removed from the preserved intestinal contents and counted. The length of each worm was obtained by projecting on a photographic plate the worm's image enlarged six times. Pencil tracings of these projected images were made on tissue paper. To obtain the correct length of the worm, the tracing was measured by a milled wheel which reduced by six times the length of these lines and gave readings in millimeters.

Comparative numbers of worms in each group were used to determine the amount of resistance of the chick.

EXPERIMENTAL RESULTS

Experiment 1

Fifty Single Comb White Leghorn female chicks were obtained for this experiment. At the age of 10 days the chicks were weighed and divided into two groups of approximately equal weights and the injections of the hormone to the chicks of Group I were begun as described above.

To reduce or eliminate any error due to the handling of one group of chickens more than the other, the chickens of Group II were given injections of pure vegetable oil in the same amount (0.1 cc) as those of female sex hormone in Group I.

At the age of 14 days, each chick was parasitized with

200±10 embryonated, *A. galli* eggs. Three weeks later the chickens were killed, the intestine of each removed, the contents flushed into a jar and the worms counted and measured.

As shown in Table 1 the chickens of Group I had a total of 83 worms while those of Group II had 241 worms. It was found that Group I had an average of 3.3 worms per chicken as compared with an average of 9.6 worms per chicken in Group II. The largest number of worms recovered from any chicken in Group I was 19 while the lowest was one. In Group II, 42 was the highest number of worms recovered from any one chicken while the lowest was one. Nine chickens in the injected group (I) had eliminated all of their *A. galli*, while but three of the controls (Group II) were without worms.

The average length of worms for Group I was 18.0 mm with a range of 6.7 to 28.1 mm. In Group II the average worm length was 16.1 mm with a range of 3.3 to 24.9 mm.

Using numbers of worms as a basis for determining the resistance of chickens to these parasites it can be seen that Group I with an average of 3.3 worms per chicken was much more resistant than Group II which had an average of 9.6 worms per chicken. It might appear that the chickens of Group I with an average worm length of 18.0 mm were less resistant than those of Group II which had an average worm length of 16.1 mm but this small difference falls within the range of experimental error.

Statistical treatment of the data on numbers of worms showed that there was significantly less variability in the worms of

Table 1. Comparison of numbers and total and average lengths of worms from chickens of Groups I and II in Experiment 1.

Group I. Hormone injected group				Group II. Control group			
Chick	Worms			Chick	Worms		
number	Number	Total length (mm)	Average length (mm)	number	Number	Total length (mm)	Average length (mm)
A3005	10	205.6	20.6	A3017	2	36.5	18.3
A3006	8	199.0	24.9	A3027	1	15.0	15.0
A3007	0	—	—	A3030	3	56.3	18.8
A3008	2	48.1	24.1	A3031	2	28.5	14.3
A3009	1	17.5	17.5	A3032	1	3.5	3.5
A3010	2	41.6	20.8	A3033	8	141.8	17.7
A3011	7	94.9	13.6	A3035	18	253.2	14.1
A3012	0	—	—	A3037	16	316.1	19.8
A3013	19	251.3	13.2	A3038	3	39.8	13.3
A3014	0	—	—	A3039	23	393.7	17.1
A3015	0	—	—	A3040	18	233.2	13.0
A3016	0	—	—	A3041	1	18.9	18.9
A3018	4	86.5	21.6	A3042	1	3.6	3.6
A3019	3	33.3	11.1	A3043	12	168.6	14.1
A3020	4	72.4	18.1	A3044	42	676.0	16.0
A3021	0	—	—	A3045	0	—	—
A3022	5	107.6	21.5	A3046	5	82.4	16.5
A3023	1	15.7	15.7	A3047	2	37.8	18.9
A3024	0	—	—	A3048	6	113.6	18.9
A3025	8	187.9	23.5	A3049	9	152.1	16.9
A3026	0	—	—	A3050	32	503.2	15.7
A3028	4	93.6	23.4	A3051	0	—	—
A3029	4	82.0	20.5	A3052	1	19.0	19.0
A3034	0	—	—	A3053	35	590.3	16.9
A3036	1	20.8	20.8	A3054	0	—	—
Total	83	1496.3		Total	241	3883.6	
Average	3.3	18.0		Average	9.6	16.1	

Group I than in those of Group II. The mean level of _A. galli_ in Group I also was significantly less than that of the worms in Group II.

Experiment 2

The pullets used in this experiment were Single Comb White Leghorns obtained from the same commercial hatchery as the chickens in Experiment 1. When 10 days of age the chicks were banded and grouped according to weights into two groups. Group I contained 21 chicks and Group II, 20 chicks. At this time the birds in Group I were given the first injection of female sex hormone at the rate of 0.1 cc of a 10 mg per cc concentration. Injections were continued every 2nd day excluding Sundays until a total of 10 injections had been administered. The pullets of both groups were parasitized at the age of 12 days with 200±10 embryonated _A. galli_ eggs. Three weeks after they were parasitized, the pullets were killed, the intestine of each removed and the contents flushed into a jar.

Group I had a total of 609 worms (Table 2) with a range of 1 to 84 worms. A total of 679 worms was recovered from Group II, the infections ranging from 1 to 97 worms. An average of 29.0 worms per chicken was recovered from Group I as compared with an average of 34.0 worms from Group II. One pullet of Group I had eliminated all of its worms whereas, none of those in Group II was without _A. galli_.

Table 2. Comparison of numbers and total and average lengths of worms from chickens of Groups I and II in Experiment 2.

Group I. Hormone injected group				Group II. Control group			
Chick number	Worms			Chick number	Worms		
	Number	Total length (mm)	Average length (mm)		Number	Total length (mm)	Average length (mm)
A3317	20	760.8	38.0	A3297	25	1085.3	43.4
A3318	28	1317.2	47.0	A3298	97	4326.8	44.6
A3319	28	1083.4	38.7	A3299	19	763.8	40.2
A3320	37	1386.8	37.5	A3300	80	3463.6	43.3
A3321	18	755.1	42.0	A3301	36	1435.7	39.9
A3322	32	1016.9	31.8	A3302	4	172.7	43.2
A3323	36	1343.5	37.3	A3303	31	1500.9	48.4
A3324	84	4076.6	48.5	A3304	81	3697.2	45.6
A3325	23	1065.8	46.3	A3305	9	354.9	39.4
A3326	18	798.0	44.3	A3306	77	3454.9	44.9
A3327	70	3121.3	44.6	A3307	45	2210.1	49.1
A3328	48	1936.4	40.3	A3308	50	2286.4	45.7
A3329	0	--	--	A3309	16	632.2	40.3
A3330	8	310.3	38.8	A3310	2	94.8	47.4
A3331	8	332.9	41.6	A3311	52	2076.9	39.9
A3332	1	37.3	37.3	A3312	1	27.2	27.8
A3333	1	43.2	43.2	A3313	32	1434.9	44.8
A3334	2	94.7	47.4	A3314	10	432.1	43.2
A3335	30	1449.8	48.3	A3315	8	401.7	50.2
A3336	36	1589.7	44.2	A3316	4	162.2	40.5
A3337	81	3531.2	43.6				
Total	609	26,071.0		Total	679	30,034.1	
Average	29	42.8		Average	34	44.2	

Group I had an average worm length of 42.8 mm with lengths ranging from 12.3 to 65.3 mm. The average worm length for Group II was 44.2 mm with a range of 9.5 to 69.7 mm.

As in Experiment 1, worm numbers were used again as a basis for determining the amount of resistance exhibited by the pullets. Although the difference was not as striking as in Experiment 1, Group I with an average of 29.0 worms per chicken indicated a trend toward higher resistance to the worms than did the 34.0 worm average per chicken for Group II. With regard to the lengths of worms the situation is similar to that in Experiment 1, in that the slight average difference is within the range of experimental error.

When the data on numbers of worms were analyzed statistically, it was found that the difference was not significant, but that the difference which did occur (Group I had less average worms per chicken than did Group II) was in the same direction as that in Experiment 1.

Experiment 3

This experiment was begun with 50 Single Comb White Leghorn pullets obtained from the same commercial hatchery as those used in the two earlier experiments. At the age of 10 days they were weighed and divided into two equal groups by weight and the first injections of female sex hormone begun for Group I. At two weeks of age, the pullets were parasitized with 200±10 A. galli eggs.

Three weeks later when the injections had been completed the chickens of both groups were killed, the intestines removed and contents of each flushed into a separate jar.

The results of Experiment 3, while not as striking as those of Experiment 1, showed the same general trends (Table 3). Using numbers of worms as a criterion for determining resistance, Group I with an average of 4.1 worms per chicken was more resistant to the A. galli than Group II which had an average of 6.6 worms per chicken. The difference of 2.5 worms per chicken between the groups indicated an increase in resistance in Group I due to the female sex hormone injections. Numbers of worms in Group I ranged from a low of 1 to a high of 36. A similar comparison of Group II showed a low of 1 worm and a high of 74.

Group I contained seven chickens which had eliminated all of their worms while Group II included but three fowls without worms.

Concerning the growth of A. galli, Group I had a range in length of 7.7 to 46.8 mm with an average worm length of 27.1 mm. In Group II the range was from 6.0 to 39.5 mm with an average worm length of 26.0 mm. Here again the slight difference in average worm lengths of the two groups was not significant.

Statistical analysis of the results from numbers of worms in this experiment showed significantly less variability in Group I than in Group II. The mean level in Group I was just slightly less than that in Group II.

Table 3. Comparison of numbers and total and average lengths of worms from chickens of Groups I and II in Experiment 3.

Group I. Hormone injected group				Group II. Control group			
Chick number	Worms			Chick number	Worms		
	Number	Total length (mm)	Average length (mm)		Number	Total length (mm)	Average length (mm)
A3493	5	123.7	24.7	A3518	74	1912.0	25.8
A3494	5	132.8	26.6	A3519	9	194.7	21.6
A3495	0	--	--	A3520	3	62.2	20.7
A3497	2	41.1	20.6	A3521	1	22.7	22.7
A3498	6	154.7	25.8	A3522	4	83.2	20.8
A3499	11	189.2	18.9	A3523	8	143.9	18.0
A3500	0	--	--	A3524	3	59.0	19.7
A3501	0	--	--	A3525	6	181.5	30.3
A3502	4	97.7	24.4	A3526	1	6.8	6.8
A3503	3	67.8	22.6	A3527	4	93.1	23.3
A3504	1	17.8	17.8	A3528	4	91.8	23.0
A3505	0	--	--	A3529	1	24.5	24.5
A3506	0	--	--	A3530	2	59.8	29.9
A3507	0	--	--	A3531	14	351.1	25.1
A3508	2	30.9	15.5	A3532	3	64.6	21.5
A3509	0	--	--	A3533	11	308.3	28.0
A3510	6	103.6	17.3	A3534	0	--	--
A3511	36	1357.7	36.7	A3535	5	139.7	27.9
A3512	2	43.2	21.6	A3536	1	14.5	14.5
A3513	1	17.2	17.2	A3537	2	79.3	39.7
A3514	8	166.9	20.9	A3538	1	26.2	26.2
A3515	2	52.2	26.1	A3539	3	79.7	26.6
A3516	1	28.7	28.7	A3540	6	146.1	24.4
A3517	4	59.5	14.9	A3541	0	--	--
				A3542	0	--	--
Total	99	2684.7		Total	166	4144.7	
Average	4.1	27.1		Average	6.6	25.0	

Combined Results

The combined results of the three experiments are recorded in Table 4. Seventy pullets were used respectively in Group I (injected group) and Group II (control group). Concerning the numbers of worms, Group I yielded a total of 791 worms with an average of 11.3 worms per chicken. Comparative results of Group II showed a total of 1086 worms with an average of 15.5 worms per chicken. The difference of 4.2 worms per chicken between Groups I and II gives evidence of increased resistance of the pullets to _A. galli_ in Group I due to the injections of female sex hormone.

When the numbers of worms were analyzed statistically, Group I showed significantly less variability than Group II. The mean level also was less in Group I than in Group II.

The average worm length in the combined Group I experiments was 38.2 mm as compared with the average worm length of 35.0 mm for the Group II pullets. The difference of 3.2 mm is within the limits of experimental error.

The combined data indicate that young female chickens injected intramuscularly with diethylstilbestrol are more resistant to _A. galli_ than are young female chickens which have not received the injections of this female sex hormone.

Table 4. Summary of numbers of chickens, numbers of worms and total lengths of worms in Experiments 1, 2, and 3.

Experiment	Group I Number of chicks	Group I Number of worms	Group I Total length (worms, mm)	Group II Number of chicks	Group II Number of worms	Group II Total length (worms, mm)
1	25	83	1496.3	25	241	3883.6
2	21	609	26071.0	20	679	30034.1
3	24	99	2684.7	25	166	4144.7
Total	70	791	30252.0	70	1086	38062.4
Average		11.3	38.2		15.5	35.0

DISCUSSION

The behavior of the Group I chickens which were injected with the female sex hormone was different from that of the controls. The young fowls in Group I were more placid than those of Group II. After about three injections (one week) of the hormone, the pullets manifested the secondary sexual characteristic of squatting for coverage by a male when a person's hand touched the back of the pullet.

Another secondary sexual characteristic manifested by the pullets of Group I was their greatly enlarged oviducts which resulted from injections of this female sex hormone. This supports the findings of Herrick (1944).

Statistical analysis of the data from these experiments was made to determine if any correlation existed between the average worm length and the number of worms in each group. A scatter diagram was made by plotting the average worm length for each chick against the number of worms for the same chick. As can be seen in Fig. 1, there was no distinct pattern or trend to the points on the diagram. It must be noted that these results were figured on the basis of one group of young chickens in one experiment.

In Fig. 2, the average worm length of each group in each experiment is plotted against the average number of worms in each group in each experiment. It can be seen from this figure that there is a correlation between average worm length and

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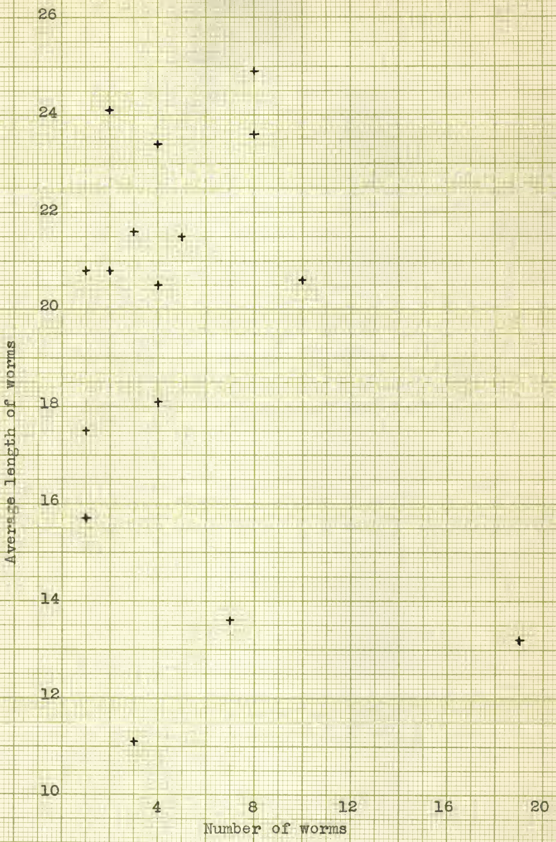

Fig. 1. Scatter diagram plotting average length and numbers of worms in Group I, Experiment 1.

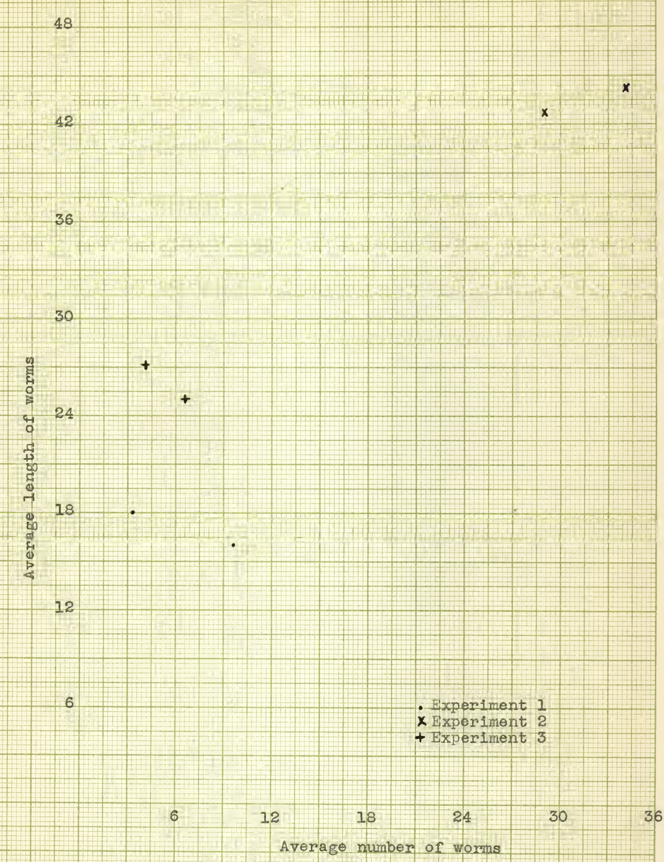

Fig. 2. Showing correlation between average worm length and average worm numbers in Experiments 1, 2 and 3.

number of worms. This correlation, however, is between experiments and not within the same experiment; hence it must be attributed to some variable which was introduced into Experiment 2, since the patterns of Experiments 1 and 3 coincide quite closely. This variable could be the inadvertent feeding of more _A. galli_ eggs to the controls than to the injected fowls of Group I.

A statistical analysis of variance showed that there was more variation in worm length from chick to chick than there was within a chick.

Statistical analysis of the data in Table 1 showed significantly less variability in the numbers of worms in Group I than in those of Group II. Also the mean level of the number of _A. galli_ from the chickens of Group I was significantly less than that of the worms in Group II. These trends were evident in the data on numbers of worms in Experiment 3 which was conducted in a manner similar to that of Experiment 1.

SUMMARY

1. A series of three experiments were performed on 140 pullets to observe if injections of female sex hormone, diethylstilbestrol, will increase the resistance of chickens to the nematode _Ascaridia galli_.

2. The criteria for determining the degree of resistance of the pullets to the _Ascaridia galli_ were the numbers and

lengths of worms harbored by the fowls.

3. The pullets of each experiment were divided into two approximately equal groups by weight. (Group I, injected; Group II, controls.)

4. Tri-weekly injections of the female sex hormone were administered intramuscularly to Group I until a total of 10 injections had been given.

5. Chickens in both groups were parasitized with 200±10 viable _Ascaridia galli_ eggs.

6. The injections of the female sex hormone produced a secondary sexual behavior pattern and a greatly enlarged oviduct in each fowl; whereas, the controls exhibited normal behavior for their age and had no enlarged oviducts.

7. Three weeks after parasitizing, the chickens were killed and the worms collected.

8. Group I had an average of 11.3 worms per chicken as compared with an average of 15.5 worms per fowl in Group II.

9. The average worm length in Group I was 33.2 mm as compared with 35.0 mm in Group II, a difference which was not significant.

10. Statistical analysis of the data on numbers of worms remaining in the chickens at the close of the experiments indicates that injections of the female sex hormone, diethylstilbestrol, increased the resistance of young female chickens to the nematode _Ascaridia galli_.

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